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Errata

Complexity in the Wiring and Regulation of Plant Circadian Networks

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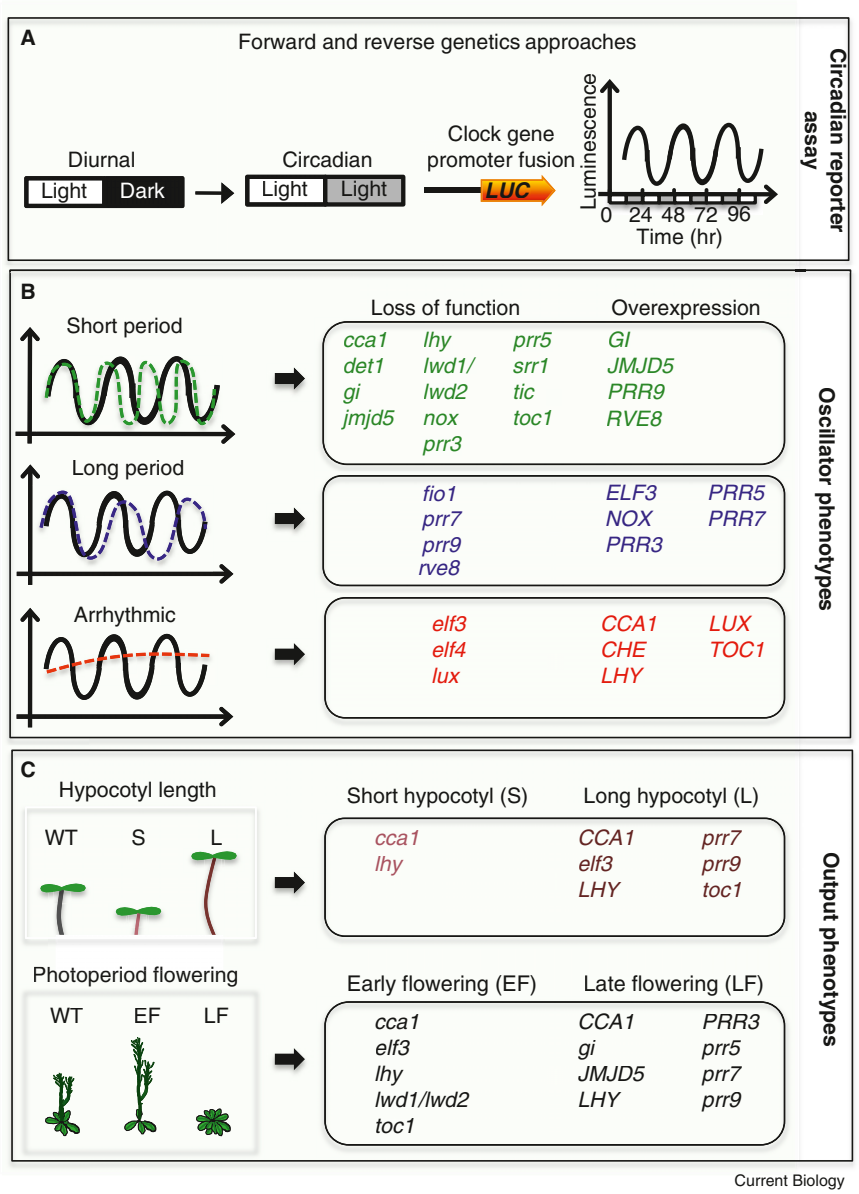
As a result of an author oversight, this Review contained several errors in Figures 1 and 2 and the Figure 2 legend. In Figure 1B, depicting long- and short-period circadian clock component phenotypes, *RVE8* is shown as long period and *rve8* as short period; this association is reversed. Instead, *RVE8* overexpression results in a short-period phenotype, whereas *rve8* (loss of function) results in a long-period phenotype. The corrected Figure 1 is shown here.



In Figure 2, JMJD5 is depicted as a direct repressor of CCA1 and LHY. The published literature does not provide sufficient data to support this interaction; JMJD5 has been removed from the corrected Figure 2 shown here. The corrected Figure 2 legend shown here has also been updated to reflect this change, as follows: "For simplification, other components that affect clock function, such as JMJD5 (also known as JMJ30), PRR3, TIC, and SRR1, are not illustrated in the figure above. Of these, the most

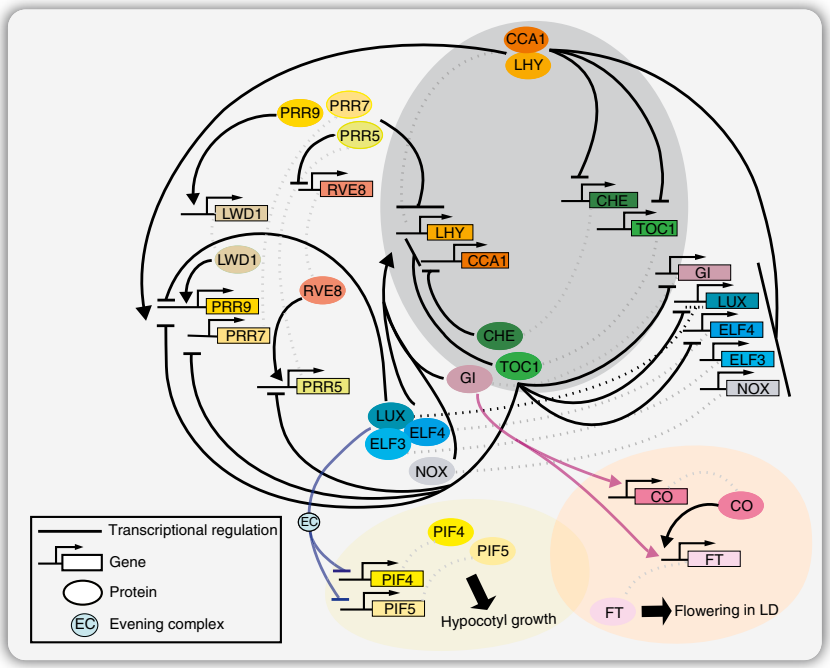


Figure 2. A Model for Transcription-Based Interactions in the *Arabidopsis* Clock Network

In vitro and in vivo assays were instrumental in validating direct molecular interactions between oscillator components. The core of the oscillator consists of two Myb transcription factors, CIRCADIAN CLOCK ASSOCIATED 1 (CCA1) and LATE ELONGATED HYPOCOTYL (LHY), and TIMING OF CAB EXPRESSION 1 (TOC1). Other components expressed throughout the day interconnect with the core oscillator to form multiple feedback loops and a complex clock network. CCA1 and LHY directly repress *TOC1*, *LUX*, *GI*, *ELF3*, *ELF4*, *CHE*, and *NOX* (also known as *BROTHER OF LUX ARRHYTHMO*) by binding to their promoters. In return, TOC1, LUX, GI, and ELF3 positively regulate *CCA1* and *LHY* via an unknown mechanism. NOX directly activates *CCA1* by binding to its promoter. LUX binds to its promoter and represses its own expression (indicated by black dashed lines). CHE functions as a direct repressor of *CCA1*. TOC1 inhibits the expression of *CCA1*, *LHY*, *PRR9*, *PRR7*, *PRR5*, *LUX*, *ELF4*, and *GI*. Sequential expression of PRR9, PRR7, and PRR5 directly inhibits the expression of *CCA1* and *LHY*. In turn, *PRR9* and *PRR7* are positively regulated by CCA1 and LHY. *PRR9* and *PRR5* are also positively regulated by LWD1 and PRR5, respectively. For simplification, other components that affect clock function, such as JMJD5 (also known as JMJ30), PRR3, TIC, and SRR1, are not illustrated in the figure above. Of these, the most recently identified component, *JMJD5*, is directly repressed by CCA and LHY, interacts genetically with TOC1, and is in turn proposed to positively regulate *CCA1* and *LHY*. Though not illustrated in the above figure, protein-protein interactions often occur between clock components and are an important mechanism regulating clock function. CCA1 and LHY physically interact. TOC1 interacts with CHE and PRR5. LUX interacts with ELF3 and ELF4 to form the evening complex (EC). Direct mechanistic connections exist between clock components and modulators of physiological processes. The EC regulates hypocotyl growth by directly binding to the promoters of *PIF4* and *PIF5*. Direct interaction between GI, *CO*, and *FT*, and between GI and *FT*, modulates photoperiod flowering. Arrows represents transcriptional activation, and horizontal lines represent repression. Dashed lines in gray indicate the protein and gene associations.

recently identified component, *JMJD5*, is directly repressed by CCA and LHY, interacts genetically with TOC1, and is in turn proposed to positively regulate *CCA1* and *LHY*."

The authors apologize for any confusion that may have resulted from these errors.

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